

WEST Search History

DATE: Monday, July 07, 2003

Set Name Query
side by side

Hit Count Set Name
result set

DB=USPT; PLUR=YES; OP=OR

L35	L34 and (clot\$ or coagul\$ or anticoagul\$ or anti-coagul\$ or "anti coagulant" or "anti coagulation")	36	L35
L34	l16 and t-pa same aprotinin\$	36	L34
L33	l16 same t-pa same aprotinin\$	0	L33
L32	l16 same t-pa	193	L32
L31	l28 same (clot\$ or coagul\$ or anticoagul\$ or anti-coagul\$ or "anti coagulant" or "anti coagulation")	25	L31
L30	L28 same l16	6	L30
L29	L28 and l16	196	L29
L28	"receptor associated protein" or rap	1670	L28
L27	l16 same l17 same (clot\$ or coagul\$ or anticoagul\$ or anti-coagul\$ or "anti coagulant" or "anti coagulation")	1	L27
L26	L25 and (clot\$ or coagul\$ or anticoagul\$ or anti-coagul\$ or "anti coagulant" or "anti coagulation")	100	L26
L25	L24 or l21 or l23	143	L25
L24	l16 and ("tissue type plasminogen activator" or tpa?) same aprotinin\$	5	L24
L23	l16 and ("tissue type plasminogen activator" or tpa?) and aprotinin\$	105	L23
L22	l16 same ("tissue type plasminogen activator" or tpa?) same aprotinin\$	0	L22
L21	l16 same l17	38	L21
L20	l16 and L19	2000	L20
L19	L18 or l17	9819	L19
L18	("tissue type plasminogen activator" or tpa?) and aprotinin\$	206	L18
L17	"receptor associated protein" or rap?	9614	L17
L16	(Fviii\$ or "factor viii" or viii\$ or "von willebrand factor" or vwf or fv or "factor v")	127960	L16
L15	(jeb or bullosa\$) same (1368 or 476)	0	L15
L14	L12 and (jeb or eb)	3	L14
L13	L12 and bullosa?	0	L13
L12	(476) and "termination codon" and (bullosa? or g2 or gamma2? or lamc2 or laminin-5 or "laminin 5")	30	L12
L11	(476) and codon and (bullosa? or g2 or gamma2? or lamc2 or laminin-5 or "laminin 5")	171	L11
L10	(1368 or 1368insc?) same laminin\$	1	L10

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009999...Open

DIALOG INFORMATION SERVICES

PLEASE LOGON:

ENTER PASSWORD:

Welcome to DIALOG

Dialog level 02.16.02D

Last logoff: 07Jul03 11:41:03

Logon file405 07Jul03 15:30:53

* * * * See HELP NEWS 225 for information on new search prefixes
and display codes

SYSTEM:HOME

Cost is in DialUnits

Menu System II: D2 version 1.7.9 term=ASCII

*** DIALOG HOMEBASE(SM) Main Menu ***

Information:

1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:

6. DIALOG(R) Document Delivery
7. Data Star(R)

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/H = Help /L = Logoff /NOMENU = Command Mode

Enter an option number to view information or to connect to an online service. Enter a BEGIN command plus a file number to search a database (e.g., B1 for ERIC).

? b 410

07Jul03 15:30:53 User268147 Session D104.1
\$0.00 0.151 DialUnits FileHomeBase
\$0.00 Estimated cost FileHomeBase
\$0.00 Estimated cost this search
\$0.00 Estimated total session cost 0.151 DialUnits

File 410:Chronolog(R) 1981-2003/Aug

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Set Items Description

? set hi %%%;set hi %%

HIGHLIGHT set on as "
HIGHLIGHT set on as "
? b 5, 34, 155, 172
07jul03 15:31:00 User268147 Session D104.2
\$0.00 0.072 DialUnits File410
\$0.00 Estimated cost File410
\$0.01 TELNET
\$0.01 Estimated cost this search
\$0.01 Estimated total session cost 0.223 DialUnits

SYSTEM:OS - DIALOG OneSearch
File 5:Biosis Previews(R) 1969-2003/Jun W5
(c) 2003 BIOSIS
File 34:SciSearch(R) Cited Ref Sci 1990-2003/Jun W5
(c) 2003 Inst for Sci Info
File 155:MEDLINE(R) 1966-2003/Jun W5
(c) format only 2003 The Dialog Corp.
*File 155: Medline has been reloaded and accession numbers have changed. Please see HELP NEWS 155.
File 172:EMBASE Alert 2003/Jul W1
(c) 2003 Elsevier Science B.V.

Set Items Description

? s fviii? or "factor viii" or viii? or "von villebrand factor" or vwf or fv or "factor v"
4434 FVIII?

? ds

Set Items Description
S1 79094 FVIII? OR "FACTOR VIII" OR VIII? OR "VON VILLEBRAND FACTOR"
OR VWF OR FV OR "FACTOR V"
S2 5440 "RECEPTOR ASSOCIATED PROTEIN" OR "RECEPTOR ASSOCIATED PROT-
EINS" OR RAP
S3 110 ("TISSUE TYPE PLASMINOGEN ACTIVATOR" OR "TISSUE TYPE PLASM-
INOGEN ACTIVATORS" OR TPA? OR T-PA) AND APROTININ?
S4 273394 CLOT OR CLOTTING OR CLOTS OR COAGULANT? OR COAGULATION? OR
ANTICOAGULANT? OR ANTI-COAGULANT? OR ANTI-COAGULAT\$

? ds

Set Items Description
S1 79094 FVIII? OR "FACTOR VIII" OR VIII? OR "VON VILLEBRAND FACTOR"
OR VWF OR FV OR "FACTOR V"
S2 5440 "RECEPTOR ASSOCIATED PROTEIN" OR "RECEPTOR ASSOCIATED PROT-
EINS" OR RAP
S3 110 ("TISSUE TYPE PLASMINOGEN ACTIVATOR" OR "TISSUE TYPE PLASM-
INOGEN ACTIVATORS" OR TPA? OR T-PA) AND APROTININ?
S4 273394 CLOT OR CLOTTING OR CLOTS OR COAGULANT? OR COAGULATION? OR
ANTICOAGULANT? OR ANTI-COAGULANT? OR ANTI-COAGULAT\$

? s s1 and s2

79094 S1

5440 S2

S5 23 S1 AND S2

? s s1 and s3

79094 S1

110 S3

S6 5 S1 AND S3

? s s5 or s6

23 S5

5 S6

S7 28 S5 OR S6

? s py<=1999

Processing
Processing
Processing
Processing
S831283591 PY<=1999

? s s7 and s8

28 S7

31283591 S8

S9 19 S7 AND S8

? type s9/full/all

9/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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12260190 BIOSIS NO.: 200000013692

The second and fourth cluster of class A cysteine-rich repeats of the low density lipoprotein receptor-related protein share ligand-binding properties.

AUTHOR: Neels Jaap G(a); van den Berg Birgit M M; Lookene Aivar; Olivecrona Gunilla; Pannekoek Hans; van Zonneveld Anton-Jan

AUTHOR ADDRESS: (a)Department of Biochemistry, Academic Medical Center, University of Amsterdam, Meibergdreef 15, 1105 AZ, Amsterdam**Netherlands

JOURNAL: Journal of Biological Chemistry 274 (44):p31305-31311 Oct. 29, 1999

ISSN: 0021-9258

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: The low density lipoprotein receptor-related protein (LRP) is a multifunctional endocytic cell-surface receptor that binds and internalizes a diverse array of ligands. The receptor contains four putative ligand-binding domains, generally referred to as clusters I, II, III, and IV. In this study, soluble recombinant receptor fragments, representing each of the four individual clusters, were used to map the binding sites of a set of structurally and functionally distinct ligands. Using surface plasmon resonance, we studied the binding of these fragments to methylamine-activated alpha2-macroglobulin, pro-urokinase-type plasminogen activator, tissue-type plasminogen activator (t-PA), plasminogen activator inhibitor-1, t-PAcndotplasminogen activator inhibitor-1 complexes, lipoprotein lipase, apolipoprotein E, tissue factor pathway inhibitor, lactoferrin, the light chain of blood coagulation factor VIII, and the intracellular chaperone receptor-associated protein (RAP). No binding of the cluster I fragment to any of the tested ligands was observed. The cluster III fragment only bound to the anti-LRP monoclonal antibody alpha2M α 3 and weakly to RAP. Except for t-PA, we found that each of the ligands tested binds both to cluster II and to cluster IV. The affinity rate constants of ligand binding to clusters II and IV and to LRP were measured, showing that clusters II and IV display only minor differences in ligand-binding kinetics. Furthermore, we demonstrate that the subdomains C3-C7 of cluster II are essential for binding of ligands and that this segment partially overlaps with a RAP-binding site on cluster II. Finally, we show that one RAP molecule can bind to different clusters simultaneously, supporting a model in which RAP binding to LRP induces a conformational change in the receptor that is incompatible with ligand binding.

08315273 Genuine Article#: 270GW Number of References: 57
Title: Role of the low density lipoprotein-related protein receptor in mediation of factor VIII catabolism
Author(s): Saenko EL (REPRINT) ; Yakhyaev AV; Mikhailenko I; Strickland DK; Sarafanov AG
Corporate Source: AMER RED CROSS,HOLLAND LAB, 15601 CRABBS BRANCH WAY/ROCKVILLE/MD/20855 (REPRINT)
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1999, V274, N53 (DEC 31), P 37685-37692
ISSN: 0021-9258 Publication date: 19991231
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY
Abstract: In the present study, we found that catabolism of coagulation factor VIII (fVIII) is mediated by the low density lipoprotein receptor-related protein (LRP), a liver multiligand endocytic receptor. In a solid phase assay, fVIII was shown to bind to LRP (K-d 116 nM). The specificity was confirmed by a complete inhibition of fVII/LRP binding by 39-kDa receptor-associated protein (RAP), an antagonist of all LRP ligands. The region of fVIII involved in its binding to LRP was localized within the A2 domain residues 484-509, based on the ability of the isolated A2 domain and the synthetic A2 domain peptide 484-509 to prevent fVIII interaction with LRP. Since vWF did not inhibit fVIII binding to LRP, we proposed that LRP receptor may internalize fVIII from its complex with vWF. Consistent with this hypothesis, mouse embryonic fibroblasts that express LRP, but not fibroblasts genetically deficient in LRP, were able to catabolize I-125-fVIII complexed with vWF, which was not internalized by the cells. These processes could be inhibited by RAP and A2 subunit of fVIII, indicating that cellular internalization and degradation were mediated by interaction of the A2 domain of fVIII with LRP. In vivo studies of I-125-fVIII vWF complex clearance in mice demonstrated that RAP completely inhibited the fast phase of the biphasic I-125-fVIII clearance that is responsible for removal of 60% of fVIII from circulation. Inhibition of the RAP-sensitive phase prolonged the half-life of I-125-fVIII in circulation by 3.3-fold, indicating that LRP receptor plays an important role in fVIII clearance.
Identifiers--KeyWord Plus(R): VON-WILLEBRAND-FACTOR; CELL-SURFACE

9/9/9 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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05562519 Genuine Article#: WG900 Number of References: 54
Title: Coagulation and fibrinolytic profile of paediatric patients undergoing cardiopulmonary bypass
Author(s): Chan AKC; Leaker M; Burrows FA; Williams WG; Gruenwald CE; Whyte L; Adams M; Brooker LA; Adams H; Mitchell L; Andrew M (REPRINT)
Journal: THROMBOSIS AND HAEMOSTASIS, 1997, V77, N2 (FEB), P270-277
ISSN: 0340-6245 Publication date: 19970200
Publisher: F K SCHATTAUER VERLAG GMBH, P O BOX 10 45 45, LENZHALDE 3, D-70040 STUTTGART, GERMANY

Language: English Document Type: ARTICLE
Geographic Location: CANADA
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: HEMATOLOGY; PERIPHERAL VASCULAR DISEASE
Abstract: The haemostatic system and the use of heparin during cardiopulmonary bypass (CPB) have been studied extensively in adults but not in children. Results from adult trials cannot be extrapolated to children because of age-dependent physiologic differences in haemostasis. We studied 22 consecutive paediatric patients who underwent CPB at The Hospital for Sick Children, Toronto. Fibrinogen, factors II, V, VIII, VIII, IX, XI, XII, prekallikrein, protein C, protein S, antithrombin (AT), heparin cofactor II, alpha(2)-macroglobulin, plasminogen, alpha(2)-antiplasmin, tissue plasminogen activator (tPA), plasminogen activator inhibitor, thrombin-AT complexes (TAT), D-dimer, heparin (by both anti-factor Xa assay and protamine titration) and activated clotting time (ACT) were assayed perioperatively. The timing of the sampling was: pre heparin, post heparin, after initiation of CPB, during hypothermia, post hypothermia, post protamine reversal and 24 h post CPB. Plasma concentrations of all haemostatic proteins decreased by an average of 56% immediately following the initiation of CPB due to haemodilution. During CPB, the majority of procoagulants, inhibitors and some components of the fibrinolytic system (plasminogen, alpha(2)AP) remained stable. However, plasma concentrations of TAT and D-dimers increased during CPB showing that significant activation of the coagulation and fibrinolytic systems occurred. Mechanisms responsible for the activation of haemostasis are likely complex. However, low plasma concentrations of heparin (<2.0 units/ml in 45% of patients) during CPB were likely a major contributing etiology. ACT values showed a poor correlation ($r = 0.38$) with heparin concentrations likely due to concurrent haemodilution of haemostatic factors, activation of haemostatic system, hypothermia and activation of platelets. In conclusion, CPB in paediatric patients causes global decreases of components of the coagulation and fibrinolytic systems, primarily by haemodilution and secondarily by consumption.

Identifiers--KeyWord Plus(R): CONGENITAL HEART-DISEASE; ANTITHROMBIN-III;

9/9/11 (Item 6 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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04295482 Genuine Article#: RU672 Number of References: 69
Title: FIBRINOLYSIS INHIBITS SHEAR STRESS-INDUCED PLATELET-AGGREGATION
Author(s): KAMAT SG; MICHELSON AD; BENOIT SE; MOAKE JL; RAJASEKHAR D;
HELLUMS JD; KROLL MH; SCHAFER AI
Corporate Source: VET AFFAIRS MED CTR,MED SERV,2002 HOLCOMBE
BLVD/HOUSTON//TX/77030; VET AFFAIRS MED CTR,MED SERV/HOUSTON//TX/77030;
RICE UNIV/HOUSTON//TX/77030; UNIV MASSACHUSETTS,SCH
MED/WORCESTER//MA/00000; MED CTR CENT MASSACHUSETTS/WORCESTER//MA/00000
Journal: CIRCULATION, 1995, V92, N6 (SEP 15), P1399-1407
ISSN: 0009-7322
Language: ENGLISH Document Type: ARTICLE
Geographic Location: USA
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences; CC CLIN--
Current Contents, Clinical Medicine
Journal Subject Category: CARDIOVASCULAR SYSTEM; HEMATOLOGY
Abstract: Background Shear stress-induced platelet aggregation may initiate arterial thrombosis at sites of pathological blood flow. Shear stress-induced platelet aggregation is mediated by von Willebrand factor (vWF) binding to platelet membrane glycoprotein (GP) Ib

and GP IIb/IIIa. Tissue-type plasminogen activator (TPA) induces thrombolysis in coronary arteries through the local generation of plasmin. Plasmin also proteolyses GP Ib and plasma vWF.

Methods and Results Because these effects could mitigate shear stress-induced platelet aggregation, we investigated the effect of fibrinolytic agents on platelet aggregation in response to a pathological shear stress of 120 dynes/cm² generated by a cone-and-platen rotational viscometer. Plasmin inhibited shear stress-induced aggregation of washed platelets, and this was associated with a decrease in GP Ib. TPA, at concentrations greater than or equal to 2000 IU/mL, significantly inhibited shear stress-induced platelet aggregation of platelet-rich plasma without a decrease in platelet GP Ib. In plasma-platelet mixing experiments, we determined that the TPA effect was localized to plasma. Purified vWF multimer degradation by TPA (in the presence of exogenous plasminogen) was associated with the loss of the capacity of vWF to support shear stress-induced platelet aggregation.

Conclusions These results demonstrate that TPA inhibits platelet aggregation in response to pathological shear stress by altering the multimeric composition of vWF. This effect of TPA on shear stress-induced platelet aggregation may contribute, along with fibrinolysis, to the therapeutic effect of TPA in restoring blood flow during acute coronary artery thrombosis.

Descriptors--Author Keywords: STRESS, SHEAR ; ENZYMES ; PLASMINOGEN

WION KL, 1985, V317, P726, NATURE

9/9/13 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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01370113 Genuine Article#: GT872 Number of References: 25
Title: EFFECTS ON COAGULATION AND FIBRINOLYSIS OF DESMOPRESSIN IN PATIENTS
UNDERGOING TOTAL HIP-REPLACEMENT
Author(s): FLORDAL PA; LJUNGSTROM KG; SVENSSON J; EKMAN B; NEANDER G
Corporate Source: DANDERYD HOSP,KAROLINSKA INST,DEPT
SURG/S-18288DANDERYD//SWEDEN/; DANDERYD HOSP,DEPT CLIN CHEM/S-18288
DANDERYD//SWEDEN/; DANDERYD HOSP,DEPT ANAESTHESIOL/S-18288
DANDERYD//SWEDEN/; DANDERYD HOSP,DEPT ORTHOPED/S-18288
DANDERYD//SWEDEN/
Journal: THROMBOSIS AND HAEMOSTASIS, 1991, V66, N6, P652-656
Language: ENGLISH Document Type: ARTICLE
Geographic Location: SWEDEN
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: HEMATOLOGY; CARDIOVASCULAR SYSTEM
Abstract: Twelve patients undergoing total hip replacement, with regional anaesthesia and with dextran infusion for plasma expansion and thromboprophylaxis, were given the vasopressin analogue desmopressin (DDAVP) or placebo in a randomized, double-blind prospective study. In controls (n = 6) we found a prolongation of the bleeding time, low factor VIII (FVIII) and von Willebrand factor (vWF) and a decrease in antithrombin III to levels known to be at risk for venous thrombosis. Desmopressin shortened postoperative bleeding time, gave an early FVIII/vWF complex increase, prevented antithrombin III from falling to critically low values and appeared to activate the fibrinolytic system, both by tPA increase and PAI-1 decrease.

Thus in the controls we found changes in both coagulation and

fibrinolysis indicating a haemorrhagic diathesis as well as a risk for thromboembolism. Desmopressin induced factor changes that possibly reduce both risks.

Identifiers--KeyWords Plus: DEEP-VEIN THROMBOSIS; BLOOD-LOSS; GENERAL-ANESTHESIA; SURGERY; VASOPRESSIN; TRIAL

9/9/14 (Item 9 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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01029333 Genuine Article#: FP706 Number of References: 30

Title: EFFECTS OF DESMOPRESSIN AND DEXTRAN ON COAGULATION AND FIBRINOLYSIS IN HEALTHY-VOLUNTEERS

Author(s): FLORDAL PA; SVENSSON J; LJUNGSTROM KG

Corporate Source: DANDERYD HOSP,DEPT SURG/S-18288 DANDERYD//SWEDEN/;
DANDERYD HOSP,DEPT CLIN CHEM/S-18288 DANDERYD//SWEDEN/

Journal: THROMBOSIS RESEARCH, 1991, V62, N5, P355-364

Language: ENGLISH Document Type: ARTICLE

Geographic Location: SWEDEN

Subfile: SciSearch; CC LIFE-Current Contents, Life Sciences

Journal Subject Category: CARDIOVASCULAR SYSTEM; HEMATOLOGY

Abstract: The effects of desmopressin and dextran on haemostasis and fibrinolysis were studied in four healthy volunteers. Both drugs were compared to placebo, each volunteer being subject to four experiments. Dextran 70 (30 g i.v.) moderately decreased VIII:C and vWF :Ag and slightly increased antithrombin III, also when haemodilution and diurnal variation were considered. Desmopressin (0.3-mu-g/kg BW i.v.), alone as well as in combination with dextran, increased VIII:C, vWF:Ag, protein C and tPA and decreased PAI-1. The combination of desmopressin and dextran stimulated coagulation and fibrinolysis and might be of relevance to surgical blood loss as well as to postoperative thromboembolism.

Descriptors--Author Keywords: DESMOPRESSIN; DEXTRANS; COAGULATION; FIBRINOLYSIS

Identifiers--KeyWords Plus: ARGININE VASOPRESSIN DDAVP; VENOUS THROMBOSIS;

9/9/15 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)
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11801593 99240769 PMID: 10224142

Rap1B and Rap2B translocation to the cytoskeleton by von Willebrand factor involves FcgammaII receptor-mediated protein tyrosine phosphorylation.

Torti M; Bertoni A; Canobbio I; Sinigaglia F; Lapetina E G; Balduini C
Department of Biochemistry, University of Pavia, via Bassi 21, 27100
Pavia, Italy. mtorti@unipv.it

Journal of biological chemistry (UNITED STATES) May 7 1999, 274
(19) p13690-7, ISSN 0021-9258 Journal Code: 2985121R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Stimulation of human platelets with von Willebrand factor (vWF) induced the translocation of the small GTPases Rap1B and Rap2B to the cytoskeleton. This effect was specifically prevented by an anti-glycoprotein Ib monoclonal antibody or by the omission of stirring, but was not affected by the peptide RGDS, which antagonizes binding of adhesive proteins to platelet integrins. Association of Rap2B with the cytoskeleton was very rapid, while translocation of Rap1B occurred in a

later phase of platelet activation and was totally inhibited by cytochalasin D. vWF also induced the rapid tyrosine phosphorylation of several proteins that was prevented by the tyrosine kinases inhibitor genistein and by cAMP-increasing agents. Under these conditions, also the association of Rap1B and Rap2B with the cytoskeleton was prevented. Translocation of Rap proteins to the cytoskeleton induced by vWF, but not by thrombin, was inhibited by a monoclonal antibody against the Fc_γII receptor. The same antibody inhibited vWF-induced tyrosine phosphorylation of selected substrates with molecular masses of about 75, 95, and 150 kDa. Three of these substrates were identified as the tyrosine kinase pp72(syk), the phospholipase C_γ2, and the inositol 5-phosphatase SHIP. Our results indicate that translocation of Rap1B and Rap2B to the cytoskeleton is regulated by tyrosine kinases and suggest a novel role for the Fc_γII receptor in the mechanism of platelet activation by vWF.

Set	Items	Description
S1	79094	FVIII? OR "FACTOR VIII" OR VIII? OR "VON VILLEBRAND FACTOR" OR VWF OR FV OR "FACTOR V"
S2	5440	"RECEPTOR ASSOCIATED PROTEIN" OR "RECEPTOR ASSOCIATED PROTEINS" OR RAP
S3	110	("TISSUE TYPE PLASMINOGEN ACTIVATOR" OR "TISSUE TYPE PLASMINOGEN ACTIVATORS" OR TPA? OR T-PA) AND APROTININ?
S4	273394	CLOT OR CLOTTING OR CLOTS OR COAGULANT? OR COAGULATION? OR ANTICOAGULANT? OR ANTI-COAGULANT? OR ANTI-COAGULAT\$
S5	23	S1 AND S2
S6	5	S1 AND S3
S7	28	S5 OR S6
S8	31283591	PY<=1999
S9	19	S7 AND S8